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WITHIN recent years there has apparently been a great increase in the number of instances of food-poisoning. This increase has been partly real and partly only apparent. The actual increase has been due to the larger consumption of preserved foods and the wider distribution of food from a given point. When we recognize the facts that the scientific principles of bacteriology are involved in the preparation of every can of preserved food and that this work is done wholly by those who are grossly ignorant of these principles, we can only wonder that harm does not come to the consumer more frequently than it does. Moreover, diseased and damaged articles of food can be worked in preserved preparations and sold as prime goods when they could not be so disposed of otherwise. We do not claim that this is a fraud frequently practised, but that it has been occasionally resorted to both in this country and in Europe has been abundantly demonstrated.

It is not our purpose in this paper to discuss the use of tuberculous meat and milk or of any other food invaded by specific infection; but we wish to give our attention to those cases of food-poisoning in which the infecting agent is a saprophytic poison-producing microorganism.

The apparent increase in the number of instances of this kind is due to the fact that the medical profession has but recently learned to recognize this form of food-infection as a cause of illness, or, at least, has been in the possession of the knowledge necessary to convert suspicion into demonstration. Only a few years ago we were seeking for the cause of the summer diarrheas of infancy in mysterious telluric and meteorologic conditions, but now we know that these diseases are solely due to infected, and consequently poisonous, food. Formerly many cases of food-poisoning were supposed to be due to the accidental or criminal addition of some metallic or vegetable poison to the food, and unjust accusation, possibly in some instances unjust punishment, has resulted. Now we know that not only the symptoms of gastrointestinal irritant, inorganic poisons, but those of typhoid fever and typhus fever, scarlet fever, and other acute exanthemata, and even those of pneumonia, may be closely simulated by the symptoms induced by infected foods.

The effects of poisonous foods as gastrointestinal irritants are too well known to demand illustrative examples. The production of a continued fever closely simulating typhoid has been repeatedly observed. The following brief abstract of a case reported by Gaffky will serve as an illustration:

D., the chemist of the Hygienic Institute at Giessen, did not feel well on the morning of October 10th, but in company with assistant B. he visited Frankfort. During the day he had a severe headache, no appetite, and frequent chilly sensations. On returning to Giessen at night he was scarcely able to walk. On the next day he refused all food, was slightly delirious, and had one watery stool. On the 13th his condition showed serious infection. His face was red, his eyes sunken, his temperature 105.2°, and he lay in a half-unconscious state. The tongue was heavily coated; the abdomen distended and painful on pressure. He had five dark-brown, later greenish, stools. The urine was concentrated and contained 2 per cent. of albumin, as determined by Esbach tubes; it gave the diazo-reaction and the indican-reaction and contained white blood-corpuscles and numerous granular casts.

From the 13th to the 17th the patient was stupid, but not delirious. The abdomen was greatly distended and from 20 to 24 stools were passed each day, with great tenesmus. The temperature remained high notwithstanding repeated one-gram doses of antipyrin. Sleep was broken and one gram of sulfonal was given at night. The urine remained as before, and the pulse varied from 92 to 100.

From the 18th to the 20th the mental dulness was less marked. Appetite was somewhat improved and the number of stools decreased to from 8 to 10 in 24 hours. The amount of albumin in the urine was somewhat decreased and microscopic examination showed fatty casts and white blood-corpuscles. Small doses of opium were given by mouth and in suppositories.

On the morning of the 21st hemorrhage from the bowels, about 300 c.c. in amount, occurred. Several doses of opium were given and ice-bags were kept on the abdomen. After the hemorrhage there

were three slightly bloody stools. From this time the improvement was slow, but fairly constant. The fever disappeared October 29th.

After recovery, marked anesthesia of the anterior surface of the thigh developed and remained for some weeks, the anesthetic area gradually becoming smaller. The hair fell out, mental activity tired, and the eyes were easily fatigued for some months. Two workers in the same laboratory were affected in the same manner, but less severely. The only food or drink which these three men had in common was some uncooked milk taken on the morning of October 9th. D. ordered the milk sent to the laboratory, drank the greater part of it himself, giving B. a small cupful, and the servant drank a little left by the others. The cow that gave this milk was suffering from a bloody diarrhea. Gaffky found in the stools of the cow and in those of the patients a small highly virulent form of the bacillus coli, and to this he ascribes the ill effects. He supposes that some of the liquid discharges from the cow fell on the udder and thus found its way into the milk.

Gaffky suggests that the epidemic in Christiana in 1888, in which 6000 persons sickened within three weeks, was probably due to milk-infection. Hausemann states that this epidemic was regarded as *morbus sui generis*. It was evidently neither cholera nor typhoid fever. Half of those affected were children, and yet nurslings escaped altogether. However, milk, as an etiologic factor, seems not to have been considered by the attending physicians.

A case of food-poisoning resembling scarlet fever was seen by one of us two years ago and may be briefly reported as follows:

K., a very vigorous man of 34, ate freely of canned salmon. Others at the table with him remarked that the taste of the salmon was peculiar and refrained from eating it. Twelve hours later K. began to suffer from nausea, vomiting, and a griping pain in the abdomen. Eighteen hours after he had eaten the fish, Dr. Vaughan saw him. He was vomiting small quantities of mucus, colored with bile, at frequent intervals. The bowels had not moved and the griping pain continued. He was covered with a scarlatinous rash from head to foot. His pulse was 140, temperature 102°, and respiration shallow and irregular. The stomach and large intestines were washed out thoroughly, and ten grains of calomel, soon followed by twelve ounces of solution of magnesium citrate, were administered, for the purpose of cleansing the small intestines. After these medicines had acted freely, K. began to improve. The next day the rash had disappeared, but the temperature remained above the normal for four or five days, and it was not until a week later that he was able to leave his house. The remainder of the salmon was submitted to various tests. The absence of inorganic poisons was demonstrated. It was found that the subcutaneous injection of twenty drops of the fluid expressed from the salmon caused evident illness and suffering in a white rat. The only germ that could be found either by direct microscopic examination or by the preparation of plate-cultures was a micrococcus, and this was present in the salmon in great numbers. This germ grew fairly well in beef-tea, but the injection of five c.cm. of beef-tea cultures of different ages failed to affect white rats, kittens, or rabbits. However, this micrococcus when grown for twenty days in a sterilized egg, after Hueppe's method of anaerobic culture, produced a most potent poison. The white

of the egg became thin, watery, markedly alkaline, and ten drops sufficed to kill white rats.

The resemblance of the symptoms of food-poisoning in some instances to epidemic pneumonia is well illustrated in the Middlesborough pneumonia-epidemic which has been reported by Ballard and is too well known to require further mention, except to state that 490 deaths resulted.

That Winckel's disease, a septic pneumonia, may be due to infection of the food with the bacillus enteritidis first discovered by Gärtner in poisonous meat has been demonstrated. Lubarsch has reported such a case in an infant.

The stools were greenish and of bad odor. The child became cyanotic and the respiration increased to sixty per minute. Auscultation and percussion were negative in results. The breathing was wholly costal and the abdomen distended. The urine contained neither albumin nor hemoglobin. Postmortem examination showed pneumonia of the left lower lobe, bilateral purulent bronchitis, atelectasis of the right lung, parenchymatous cloudiness of the kidneys, fatty infiltration and engorgement of the liver, and slightly enlarged spleen. All other pathologic conditions were believed to be consequent upon the septic pneumonia. Plates made from the diseased organs developed only the bacillus enteritidis.

It is quite impossible to draw any sharp line between intoxication and infection in the study of the symptoms of food-poisoning. Some of the bacterial poisons are slow in inducing their effects, and even when a germ-free preparation is used in our experiments upon animals, it [sometimes happens that

many hours elapse before any effects are observed, even in cases ultimately terminating fatally. The relation of the germ to the production of the untoward symptoms may, according to the case, be stated in one or the other of the following ways: (1) The chemic poison is generated only and wholly in the food before it is taken; (2) The infecting organism may begin the elaboration of its poisonous products outside, and continue the same within, the body; (3) The infection may not result in the production of poisons until the food is taken into the body. The first of these conditions is well illustrated by the "Iron Bridge" cases reported by Ballard. The infecting germ would not grow at a temperature above 86°. Of course animal inoculations with this germ were wholly without result, but cultures grown at 60° were markedly poisonous. It is probable that this condition holds good in a larger number of instances than we suspect. The second condition is the one existing in some cases of poisoning with milk and its products. However, in these the infecting microorganisms do not thrive in the body unless they be supplied with the food especially suited for them. The complete withdrawal of milk as a food leads to their speedy disappearance. The violent vomiting and purging which result are curative means and should not be checked unless there be danger of death from exhaustion. The most dangerous cases of cheese-poisoning are those in which vomiting and purging do not occur. Some months ago one of us saw some twenty persons who had been poisoned with cream. All but two were vomiting and purging

and complaining most vehemently. These were sure that they would die. The two exceptional ones were uttering no complaints. In fact, they were practically comatose, and thorough washing out of the stomach and intestines was resorted to before they realized that they were in danger.

When the third condition mentioned exists, a true infection results. Such is the case most probably in those instances already cited as resembling typhoid fever and pneumonia.

We have been frequently called upon to examine food suspected of having caused untoward effects, and the method followed in these examinations may be of some interest. Whenever there is the slightest possibility that a metallic poison may be present, tests for the same are made. This is always done with canned foods. However, when a teaspoonful or less of ice-cream causes vomiting and purging, the idea that these effects can be due to zinc sulphate dissolved in the freezer or to artificially prepared vanillin used in flavoring is simply too preposterous to be entertained by anyone acquainted with the quantities of these substances necessary to induce such effects. Ten years ago we went through with all the tests for metallic poisons in many samples of poisonous cheese and ice-cream, but we no longer waste our time in this manner.

The examination of foods for bacterial poisons cannot be made except in a properly equipped bacteriologic laboratory. It is our purpose to merely point out at this time the methods that may be followed. We take it for granted that the one who undertakes work of this kind is already familiar

with the ordinary technic of bacteriologic research. The line of procedure will vary somewhat with the kind of food to be examined, the form in which it has been prepared, and the quantity supplied the analyst. All samples should be examined with as little delay as possible after the article has become the object of suspicion. When delay is unavoidable, further bacterial growth should be retarded in the meantime as far as is possible by keeping the suspected article at a low temperature. Germs not present at the time of the supposed poisoning may be accidentally introduced, or nontoxicogenic bacteria may multiply to such an extent that the detection of the harmful organisms is rendered impossible.

As a rule, the quantity of the food supplied the analyst is not sufficient to allow of the detection or the isolation of the chemic poison directly. To try to find the poison in a few ounces of cheese or a small bit of meat by direct extraction is a task that would be undertaken only by one quite ignorant of the nature of these poisons. In all but exceptional instances in which many pounds of the food are supplied, the portion that reaches the laboratory can only be regarded as the bearer of the germ to the activity of which the poison is due. This germ must be detected, isolated, grown in pure culture, and its toxicogenic properties demonstrated upon lower animals. It should be clearly understood that the most thorough study of the morphologic characteristics of the germ and of the chemic properties of the poison will not suffice without an accompanying determination of the

toxicologic action of the cultures. The infectious nature of the bacterium should also be studied.

It should always be borne in mind that the article of food has probably been through several hands before reaching the analyst, some of which may not have been germ-free. In the examination of pieces of meat and cheese, the surface should be sterilized with a broad, heated knife or other piece of iron. It has been shown that bacteria deposited on such surfaces penetrate slowly. Then with other sterilized knives sections should be made and one or more small bits taken from the interior should be placed in sterilized bouillon. Not less than a dozen tubes should be inoculated in this way. Three of these should be grown aerobically at ordinary temperature ; three anaerobically at the same temperature ; three aerobically at 37°C. ; and three anaerobically at 37°C. It is quite essential that all these conditions of growth should be tried. Some of the toxicogenic germs grow best at relatively low temperature, from 20° to 25°C., and fail wholly to develop at 37°C. Others have their optimum growth at the last-mentioned temperature. Some develop only when freely supplied with air, and others only when the air is excluded.

In the examination of liquid and semi-liquid foods, such as milk, custard, cream, broths, and jellies, small bits or a few drops should be placed in sterilized bouillon and grown under the conditions already mentioned.

A growth having appeared in one or more of these tubes, the bacteria should be examined in

hanging drops and in stained mounts. If more than one organism be present, plate-cultures should be made and each germ should again be grown under the conditions mentioned.

The infectious character of each organism should be tested on the lower animals: (1) by feeding, (2) by subcutaneous inoculation, (3) by intraperitoneal inoculation, and (4) by intravenous inoculation. The animals generally employed in these experiments are white mice, white rats, guinea-pigs, kittens, and rabbits. A given germ may be toxicogenic to one of these animals and not to the others. Mice and kittens are specially suitable for feeding-experiments. Young kittens are quite susceptible to most of the bacterial poisons found in milk and its products. The quantity of the bouillon-culture, 24 hours old or older, first employed should be relatively large, from one to ten c.cm., according to the animal and the method of infection. If these amounts prove active, smaller quantities should be tried until the limit is reached.

Next, the action of cultures from which the bacteria have been removed by filtration through porcelain should be tested, and, if these prove active, the effect of different degrees of heat on the toxicity of the cultures should be determined.

If by the experiments already mentioned a toxicogenic germ has been discovered, its morphologic, cultural, tinctorial, and pathogenic properties may be studied as thoroughly as the investigator may desire. The study of the bacterial poison may also be carried to the same extent.

The following reports are taken as illustrations of the results obtained by the method given:

Dec. 31st, 1894, Dr. Traver, of Somerset, Mich., was called to see the family of Mr. Van Allen. He found the father, mother, and two children suffering from protracted vomiting and marked exhaustion. There was no fever and no diarrhea. Tyrotoxicon-poisoning was suspected, but inquiry showed that their supper had consisted of bread, butter, tea, dried beef, and raspberry-sauce. There was nothing in the appearance or odor of the meat to cause any suspicion. In fact, it seemed to be of exceptionally good quality. Anaerobic cultures from the interior of the meat were made and developed a bacillus, from two to three times as long as broad, taking the ordinary stains well, motile, with no spore-formation, not liquefying gelatin, but coagulating milk, growing best at the temperature of the body, but developing its poison at ordinary temperature, producing gas abundantly, and pathogenic to white rats, rabbits, and guinea-pigs. Sterilized cultures were also poisonous.

Of a large number of men at a banquet at Sturgis, Mich., April 26, 1894, everyone who ate of the pressed chicken served was made sick. Some who were not at the banquet, but who aided in preparing for it, ate small bits of the chicken, and were also sick. All were attacked, within from two to four hours after eating the chicken, with nausea, violent griping, and purging; many fainted while attempting to arise from bed.

The chickens were killed Tuesday afternoon, picked and left hanging in the market-room (not in a cooling-room) until Wednesday forenoon, when they were drawn and carried to a restaurant and here left in a warm room until Thursday morning,

when they were cooked (not very thoroughly), pressed, and served at the banquet that night. Those who ate of the unpressed chicken were also made ill.

The pressed chicken contained two microorganisms, a slender bacillus, from four to five times as long as broad, and a streptococcus. The bacillus was fatal to white rats, guinea-pigs, and rabbits when administered intraperitoneally, intravenously, and subcutaneously. The streptococcus was not fatal when given in pure cultures, but mixed cultures of the two induced death, and in these instances, when administered subcutaneously, in addition to the lesions formed after the employment of pure cultures of the bacillus, there was extensive sloughing. This bacillus is motile, takes the ordinary stains readily, and is decolorized by Gram's method. It grows very slowly at ordinary temperature and rapidly at 98°F. Of two cultures of equal age, one grown at ordinary temperature and the other at 98°F., one c.cm. of the former was necessary to induce death, while one-fourth c.cm. of the other proved fatal. The anaerobic cultures were much more powerful than the aerobic. On gelatin-plates exposed to the air the growth was slow, while on those kept in an atmosphere of hydrogen it was much more rapid. Spores could not be detected. The bacillus coagulates milk and decolorizes litmus-gelatin. On potatoes it forms a dirty, thick, slimy growth. It does not liquefy gelatin and the production of gas was not observed. Streaks on agar-agar are yellowish white, slimy, and with but little tendency to spread. One half c.cm. of a beef-tea culture heated to 140°F. for 30 minutes killed guinea-pigs. One c.cm. heated to 212° F. for 15 minutes failed to kill. Animals inoculated by the methods mentioned showed evidences of abdominal pain within from one to two

hours and several were found dead after twelve hours. The abdominal cavity was found filled with a clear fluid, the bloodvessels were much congested, and the peritoneum inflamed. In some instances a bloody fluid was found in the pleural cavity.